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EDITORIAL

LATS and the Thyroid Gland

ELSEWHERE IN THIS ISSUE OF CALIFORNIA MEDICINE, Kriss presents his views on the long-acting thyroid stimulator of Graves' disease. This long known disorder almost begged for a fresh approach when in 1956, Adams, a New Zealander out of Sir John Hercus' great thyroid school, attacked with new zeal and derouted the pituitary:thyroid axis as basis for the continuing thyroid overactivity. The actual discovery in guinea pigs involved their repeated use for economy's sake and was so fortuitous as to recall the accidental bounties that came to the Princes of Serendip.

Kriss, among other points, presents the basis for considering LATS an antibody, of lymphocytic but not of monoclonal origin. He looks upon thyroid injury as important in pathogenesis to explain release of thyroid proteins as antigens inducing LATS as antibody. McKenzie, another authority on LATS, reviewed the same topic in *Physiologic Reviews,* January, 1968. Many similarities in viewpoints of the two are apparent. The careful student of the disorder will wish to read each. Similarities in position greatly exceed differences but the latter deserve comment. Thus, Kriss may be noted, in discussing the possible role of desiccated thyroid in precipitating Graves' disease, to speculate on the absorption of antigens and the consequent inducing of LATS antibody. McKenzie calls attention to the role of thyroxine itself, possibly acting to enhance LATS production by the lymphocyte. Either speculation may serve as a reminder that there is potential danger in the administration of thyroxine (and thyroid) and that specific indication should precede prescription of either. McKenzie's view may explain why induction of a period of health, as with an antithyroid drug, favors a lasting remission of the disease.

A more serious consideration of divergent views turns on noting Kriss to postulate that antigen: antibody complexes (thyroid proteins reacting with LATS) are of pathogenic significance in the ophthalmopathy of Graves' disease. McKenzie assigns no significance to LATS in the pathogenesis of ophthalmology. Kriss cites Catz, with other evidence, to bolster his position—Catz claiming total removal of the thyroid gland to benefit serious ophthalmopathy, presumably because the antigen is eliminated. (Kriss also notes Werner's dissent from Catz.) The important point for the practicing physician is that the role of LATS in ophthalmopathy remains under dispute and the role of total thyroidectomy for the eye disease is a matter for most careful and critical clinical investigation.

Much of the needed evidence about LATS which will guide physicians in the care of patients with Graves' disease remains to be gathered despite the signal advances of the past decade. A most urgent need is an experimental model of the disorder in which both hyperthyroidism and ophthalmic involvement occur. This development would carry a strong likelihood of enabling truly rational treatment of Graves' disease.

REFERENCE

McKenzie, J. M.: Humoral factors in the pathogenesis of Graves' disease, Physiological Reviews, 48:252-310, Jan. 1968.